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Peanut consumption and cardiovascular risk

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Abstract

Objective: We evaluated the effects of peanut consumption on lipid profiles, atherogenic index of plasma (AIP) and CHD risk in hypercholesterolaemic men.
Design: Randomised crossover clinical trial.
Setting: Participants were randomly assigned to two groups. They were asked to consume peanut supplements (about 77 g) with their habitual diet for 4 weeks.
Subjects: Fifty-four hypercholesterolaemic men with total cholesterol (TC) concentrations between 200 and 350 mg/dl.
Results: Compared with the habitual diet, peanut supplementation of the habitual diet significantly reduced TC/HDL cholesterol (HDL-C) ratio (mean 1 (SE 0·3) $P = 0·001$) and LDL cholesterol (LDL-C)/HDL-C ratio (mean 0·7 (SE 0·2); $P = 0·001$). Peanut consumption increased HDL-C (mean 6·1 (SE 1·5) mg/dl; $P < 0·001$) and total antioxidant capacity (TAC) (mean 1·2 (SE 0·6) U/mL $P = 0·04$). In addition, peanut consumption significantly reduced the AIP (mean 0·1 (SE 0·03) $P = 0·01$) and CHD estimated risk over 10 years based on systolic and diastolic blood pressures (mean 1·4 % (SE 0·5 %) $P = 0·004$ and mean 2·2 % (SE 0·5 %) $P < 0·001$, respectively).
Conclusions: Short-term peanut consumption might improve lipid profiles, the AIP and CHD risk in free-living hypercholesterolaemic men.

Keywords
Peanut
Habitual diet
Coronary heart disease
Atherogenic index of plasma

Regular consumption of nuts is beneficial to cardiovascular health. Data from the Adventist Health Study and Nurses' Health Study show that higher nut consumption is associated with reduced risk of coronary artery disease[1,2]. In addition, other epidemiological studies have shown that frequent nut consumption decreases the risk of CHD, with adjusted relative risk reductions approaching 50 % in subjects who consume 4–5 servings per week than those who have little or no intake[3–4].

Peanuts are rich source of Mg, folate, fibre, α-tocopherol, Cu, arginine and resveratrol. All of these compounds have been shown to reduce CHD risk in various ways, and this suggests that peanut consumption might benefit those at risk for CHD. However, most studies to date have been performed in either healthy or hypercholesterolaemic subjects in combination with low-fat diet. O'Byrne *et al.*[5] reported a decrease of total cholesterol (TC) and LDL cholesterol (LDL-C) in hypercholesterolaemic, postmenopausal women on a low total fat, low SFA and high MUFA diet for 6 months. Kris-Etherton *et al.*[6] showed beneficial effects of peanut consumption on blood lipid concentrations when consumption was combined with a high MUFA, low SFA diet in normocholesterolaemic subjects. In both of these studies, MUFA intake largely substituted for SFA intake. This raises the question of whether the observed shifts in lipid profiles were due to the increase in dietary MUFA or to the reduction of SFA consumption, and thus the role of peanut consumption is not clear. Alper and Mattes[7] reported that a moderate increase in MUFA intake without a concomitant decrease in SFA did not appear sufficient to decrease TC and LDL-C in normocholesterolaemic individuals, and they showed that peanut consumption may have beneficial effects on diet composition even when the background diet is not controlled. In order to further define the role of peanut consumption in the reduction of CHD risk factors, we observed the lipid profiles of hypercholesterolaemic men who began to consume peanuts but made no other changes to their usual diet. We analysed the atherogenic index of plasma (AIP) for each subject, which has been defined as a novel marker of plasma atherogenicity. It increases in people at higher risk for CHD and may be an important tool for analysing the results of clinical trials[8]. In addition, we estimated CHD risk to determine the overall effect of peanut consumption in hypercholesterolaemic men[9].

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Experimental methods

The present study was conducted in Tehran, Iran, in 2006, and was approved by the Research Council and the Ethical Committee of the National Nutrition and Food Technology Research Institute of Shaheed Beheshti University of Medical Sciences, Tehran, IR of Iran.

The subjects were volunteers and were recruited to the study through announcement fliers sent to the Tehran University Hospital employees. Eligible individuals were male adults, 25–65 years of age, with TC levels of 200–350 mg/dl and mean TAG levels lower than 400 mg/dl. Exclusion criteria included acute or chronic diseases (diabetes, kidney, liver and thyroid diseases; cancer; or the presence of inflammatory or infectious disease), consumption of vitamin supplements, hormone therapy or medications that might have influenced the study variables (e.g. antihypertensive and antilipidemic agents administered in the 4 months preceeding the study), recent history of weight gain or loss (≥9 kg) within the past 6 months, very atypical diet, rigorous exercise programme, allergy or aversion to nuts, habitual consumption of more than 70 g of nuts per week, cigarette smokers and first-degree family history of CHD.

Sixty hypercholesterolaemic men met the above criteria and agreed to take part in the present study. All participants provided informed written consent. Participants were randomised between two diet sequences for 4-week periods – habitual diet and habitual diet supplemented with peanuts. Thirty participants first followed the habitual diet for 4 weeks and then supplemented their diet with peanuts for the next 4 weeks; thirty subjects followed the same diets in reverse order. Because diet-induced lipoprotein changes stabilise in <4 weeks, we incorporated a washout period of 4 weeks between diets. During the peanut supplementation period, subjects were asked to consume a daily allowance of peanuts equivalent to approximately 20 % of each subject's mean energy intake in addition to their habitual diet. Freshly roasted, lightly salted peanuts were packaged in separate bags in three different weights. Individuals whose energy intake was in the lowest tertile received 60 g of peanuts per day, those in the middle tertile recieved 77 g per day, and the highest tertile received 93 g per day. The nutrient composition of peanuts per 100 g is as follows: protein 25·7 g, fat 52·6 g and fibre 4 g. The fatty-acid composition was 21 % SFA (10·9 % of these were chains shorter than C18 carbons and 10·1 % were equal to or longer than C18), 41 % MUFA and 38 % PUFA (analysed by the Oil Seed Research and Development Company, Tehran, Iran). There was no detectable aflatoxin in the peanuts (measured by immunoaffinity column-liquid chromatography with post-column derivitisation in the Faroogh Lab, Tehran, Iran). Participants were given no specific dietary advice other than to suggest that the peanuts could be eaten with meals or as snacks. In addition, participants were given an extra coded bag of peanuts to share with family and friends to improve compliance. Compliance was assessed using 24 h diet recalls at three separate time points during the habitual diet and peanut-supplemented habitual diet periods, and, because peanuts are a particularly rich source of MUFA, serum concentration of MUFA was measured as a biological marker of adherence to the peanut-supplemented diet.

Dietary intake

Three 24 h recalls were collected by a dietitian at 1-week intervals in each diet period for all participants. The recalls were reviewed for accuracy and completeness. Food intake was calculated by converting household measures and portion sizes to grams intake. Each food item was coded according to the prescribed protocol and entered into a MSACCESS database. Energy and the nutrient content of each diet was determined using the Iranian, modified food composition table[10]. Three-day food and nutrient intakes for each period were averaged and these values were reported as the mean intake of different food groups and nutrients. To estimate the amount of energy displacement caused by the peanut supplement, we calculated displacement as follows[11]:

$$\frac{[(\text{energy in habitual diet} + \text{peanut energy}) - \text{energy in peanut-supplement diet}] \times 100}{\text{peanut energy}}$$

Measurements

The weight of each subject was measured to the nearest 100 g with a digital scale while the subjects were minimally clothed and not wearing shoes. Height was measured in a standing position, without shoes, using a tape measure while the shoulders were in a normal state. All measurements were made by the same person. The physician measured each subject's blood pressure and obtained a medical history, including detailed information about medication and nutrient supplement usage. Blood samples were obtained by venepuncture before and after each diet period. Each blood sample was taken between 07.00 and 09.00 hours after 12 h fasting period. Samples were centrifuged 30–45 min after collection. Plasma samples were stored at (−80°C) and analysed at the end of the study. TC and TAG were measured using an enzymatic colorimetric method (Pars Azmoun Co., Tehran, Iran). HDL cholesterol (HDL-C) and LDL-C were quantified by the direct method (Shanghai Rongsheng Biotech Co. Ltd, Shanghai, China). The sensitivity of the assays for TC, TAG, HDL-C and LDL-C were 3, 1, 1 and 2 mg/dl, respectively. Oxidised LDL (Ox-LDL ELISA, Mercodia AB, Uppsala, Sweden) and total antioxidant capacity (TAC) were measured with colorimetric kits (Cayman, Ann Arbor, MI, USA); the sensitivity of each of these kits was 1 mU/l and 1 U/ml, respectively. Inter- and intra-assay coefficients of variation for all tests were <10 %. AIP was calculated as the log transformation of

(TAG/HDL-C), with each value expressed in molar concentrations. Finally, CHD risk estimation is defined as the percentage likelihood of a cardiac event over a period of 10 years. We used the Cardiac Risk Assessor V98.02 software based on age, sex, systolic and diastolic blood pressures (SBP and DBP, respectively), TC and HDL-C.

Statistical analysis

The arithmetic mean value of the three diet recalls from each diet period was calculated, and a paired *t* test was used to compare results from the habitual and peanut-supplemented diet periods. To identify the effects of peanut consumption on study variables, we calculated the peanut effect with the formula $[(E_P - B_P) - (E_H - B_H)]$, where E_P is the value at the end of the peanut-supplemented diet period; B_P is the value just before the peanut-supplemented diet period; E_H is the value at the end of the habitual diet period and B_H is the value just before the habitual diet period. We performed a one-sample Student's *t* test, of which the null hypothesis is that the population means of this quantity equals zero. This equation simply shows the effects of peanut consumption on the variables. We calculated relative changes by 'Peanut effect × 100/baseline', in which 'baseline' is the average baseline value before the peanut-supplemented diet and before the habitual diet. To examine whether there was any carryover, we performed appropriate repeated-measures ANOVA. Pearson's correlation coefficient was used to assess relationships between continuous variables. Results are presented as mean values and their standard errors. All statistical analyses were performed with Statistical Package for the Social Sciences statistical software package version 10·0 (SPSS Inc., Chicago, IL, USA).

Results

Two subjects lost interest in the study within a few weeks and withdrew, and four other subjects withdrew due to unforeseen travel. Ultimately, fifty-four men completed the study. The participants had a mean age of 43 years (SE 1·3), a mean BMI of 27·5 kg/m^2 (SE 0·5) and mean serum cholesterol of 254 mg/dl (SE 4). According to participant reports, compliance with peanut ingestion was 96 %. This was supported by a 24·5 % ($P = 0·02$) increase in measured serum MUFA concentration in these subjects while they were on the peanut-supplemented diet than on their habitual diet. Daily peanut consumption was well tolerated by all subjects. The only reported side-effect was transient dizziness reported by four subjects. Dietary intake of participants during the two periods, based on the mean consumption from the three separate 24 h dietary recalls, is shown in Table 1. There were no differences in

Table 1 Mean daily intake of dietary nutrients and diet composition separated by diet periods

	Habitual diet		Peanut-supplemented diet		
	Mean	SE	Mean	SE	*P* value*
n	54·0		54·0		–
Peanut (g)	0·0		77·0	2·0	<0·001
Energy (kJ)	10 676	284	12 251	335	<0·001
Carbohydrate (% of energy)	59·0	0·7	52·0	0·7	<0·001
Protein (% of energy)	13·1	0·3	13·2	0·2	0·13
Plant protein (% of energy)	6·1	0·1	8·0	0·1	<0·001
Animal protein (% of energy)	7·0	0·3	5·2	0·3	<0·001
Total fat (% of energy)	27·9	0·6	34·8	0·6	<0·001
SFA (% of energy)	9·5	0·3	10·0	0·3	0·003
MUFA (% of energy)	11·7	0·3	14·4	0·3	<0·001
PUFA (% of energy)	6·6	0·2	10·3	0·2	<0·001
Cholesterol (mg)	250·0	12·0	222·0	15·0	0·13
Fibre (g)	21·0	0·9	29·0	1·0	<0·001
α-Tocopherol (mg)	13·7	0·5	19·8	0·6	<0·001
Folate (μg)	266·0	9·0	380·0	11·0	<0·001
Cu (mg)	1·6	0·08	2·0	0·06	<0·001
Zn (mg)	13·1	0·4	14·7	0·4	0·001
Mg (mg)	410·0	15·0	548·0	18·0	<0·001
Food groups					
Total grains (g/d)	654·0	26·0	629·0	26·0	0·27
Vegetables (g/d)	263·0	16·0	254·0	19·0	0·68
Fruits (g/d)	298·0	32·0	326·0	31·0	0·49
Meat (g/d)	118·0	8·0	109·0	9·0	0·3
Eggs (g/d)	17·0	3·0	18·0	3·0	0·95
Dairy products (g/d)	402·0	31·0	317·0	26·0	0·01
Fats (g/d)	43·0	2·0	42·0	2·0	0·57
Sweets (g/d)	13·0	3·0	11·0	2·0	0·71
Beverages (ml/d)	966·0	56·0	979·0	56·0	0·77

*P for comparison between the habitual diet and the peanut-supplemented diet by paired *t* test.

food groups consumed in the two diet periods (other than peanuts), except for dairy products.

When subjects changed from their habitual diet to the peanut-supplemented diet, the intakes of MUFA, PUFA, fibre, plant protein, α-tocopherol, Cu, folate, Zn, and Mg all significantly increased (Table 1) and the intakes of animal protein decreased (all $P<0{\cdot}001$). Body weight and blood pressure were stable throughout the study. There were no changes in TC, LDL-C or TAG; but there was a 17% increase in HDL-C and reductions in TC/HDL-C and LDL-C/HDL-C ratios (13% for both), which are shown in Table 2.

The effects of peanut consumption on biomarkers of oxidative stress, AIP and CHD risk are shown in Table 3. Peanut supplementation increased TAC of serum by 13%. In addition, peanut consumption reduced AIP by 35%. Finally, peanut consumption reduced CHD risk, which is defined as the percentage likelihood of a cardiac event over a period of 10 years by 14% and 24% based on SBP and DBP, respectively. There was no evidence of a carry-over effect between periods.

Discussion

The present study shows that the addition of peanuts to the diet, without any other dietary modification, can favourably modify lipid profiles, AIP and estimated risk of CHD in hypercholesterolaemic men.

When peanuts were consumed in addition to the subjects' usual diet, their energy and fat intake increased, but significant changes in lipid profiles occurred. TC and LDL-C did not change, but HDL-C increased significantly. This suggests that increasing MUFA and PUFA intake without decreasing SFA is not sufficient to decrease TC and LDL-C, but it can increase HDL-C and improve TC/HDL-C and LDL-C/HDL-C ratios in a cardioprotective manner[12–13]. Sheridan *et al.*[14] showed similar effects of pistachio nuts on the lipid profiles of free-living human subjects with primary, moderate hypercholesterolaemia.

TAC considers the cumulative action of all antioxidants present in plasma and body fluids and provides an integrated measurement rather than the simple sum of measurable antioxidants. Low TAC is a risk factor for ischaemic heart disease[15]. In the present study, peanut consumption increased serum TAC by 13%. In addition to TAC, the AIP was recently proposed as a marker of plasma atherogenicity, and was shown to be increased in subjects at higher risk for CHD. AIP is also inversely correlated with LDL particle size[8]. The association of TAG and HDL-C in this simple ratio theoretically reflects the balance between risk and protective lipoprotein forces. In the present study, peanut consumption reduced AIP, and this is correlated with the estimated CHD risk ($r=0{\cdot}54$, $P<0{\cdot}001$).

Estimated CHD risk is defined as the likelihood that a cardiac event will occur over a period of 10 years, and is

Table 2 Body weight, blood pressure and lipid profiles of peanut supplementation for 4 weeks in hypercholesterolaemic men

	Baseline*				Changes in							
	Habitual diet		Peanut-supplemented diet		Habitual diet		Peanut-supplemented diet		Peanut effect†		*P* values		
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	*P*‡	*P*§	*P*¶
Body weight (kg)	78·1	2·1	78·0	2·1	−0·1	0·2	0·06	0·2	0·2	0·2	0·52	0·73	0·37
Systolic blood pressure (mmHg)	120·0	1·8	120·1	2·2	−2·0	1·8	−0·2	2·0	1·9	2·7	0·26	0·93	0·5
Diastolic blood pressure (mmHg)	78·2	1·5	80·8	1·3	2·3	1·4	−0·3	1·2	−2·5	1·9	0·1	0·82	0·19
TC (mg/dl)	253·0	5·0	255·0	5·0	−3·4	4·5	6·7	4·5	10·1	6·7	0·14	0·45	0·14
LDL-C mg/dl	169·0	4·0	170·0	4·0	−0·1	3·5	6·8	3·9	7·0	4·9	0·97	0·09	0·16
HDL-C mg/dl	36·1	0·9	33·5	0·8	−1·4	0·8	4·7	0·9	6·1	1·5	0·09	<0·001	<0·001
TAG (mg/dl)	229·0	14·0	232·0	11·0	−11·4	12·8	−7·4	10·4	1·8	15·3	0·38	0·48	0·91
TC/HDL-C	7·2	0·2	7·8	0·2	0·3	0·2	−0·7	0·2	−1·0	0·3	0·16	<0·001	0·001
LDL-C/HDL-C	4·8	0·1	5·2	0·2	0·3	0·1	−0·4	0·1	−0·7	0·2	0·07	0·002	0·001

TC, total cholesterol; LDL-C, LDL cholesterol; HDL-C, HDL cholesterol.
*There was no significant difference between baseline values in the habitual diet and peanut-supplemented diet except for HDL-C.
†We calculated the peanut effect (effects of peanut supplementation) using the formula $[(E_P - B_P) - (E_H - B_H)]$, where E_P is the value at the end of the peanut-supplemented diet period and B_P is the value just before the peanut-supplemented diet period. E_H is defined as the value at the end of the habitual diet period and B_H is defined as the value just before the habitual diet period.
‡*P* values are for one-sample Student's *t* test for changes during the habitual diet period.
§*P* values are for one-sample Student's *t* test for changes during the peanut-supplemented period.
¶*P* values are for one-sample Student's *t* test of the peanut effect.

Table 3 Biomarkers of oxidative stress, atherogenic index of plasma and CHD risk assessments of peanut supplementation for 4 weeks in hypercholesterolaemic men

	Baseline*				Changes in									
	Habitual diet		Peanut-supplemented diet		Habitual diet		Peanut-supplemented diet		Peanut effect†		*P* values			
	Mean	SE	Mean	SE	Mean	SE	Mean	SE	Mean	SE	*P*‡	*P*§	*P*‖
Oxidized LDL (mU/l)	11·9	0·4	12·2	0·5	0·9	0·5	−0·2	0·5	−1·1	0·6	0·08	0·68	0·07
TAC (U/ml)	9·5	0·4	10·7	0·4	−1·0	0·4	0·3	0·5	1·2	0·6	0·02	0·6	0·04
AIP	0·41	0·03	0·46	0·03	−0·002	0·03	−0·09	0·03	−0·09	0·03	0·95	0·002	0·01
CHD risk % (based on systolic BP)	9·1	0·8	10·3	1·0	0·4	0·4	−1·2	0·4	−1·4	0·5	0·34	0·005	0·004
CHD risk % (based on diastolic BP)	9·5	0·9	11·1	1·1	0·9	0·4	−1·4	0·4	−2·2	0·5	0·01	0·001	<0·001

TAC, total antioxidant capacity; AIP, athrogenic index of plasma; BP, blood pressure.
*There was a significant difference between baseline values in the habitual diet and peanut-supplemented diet for TAC. CHD risk percentage is based on systolic BP and diastolic BP.
†We calculated the peanut effect (effects of peanut supplementation) using the formula $[(E_P - B_P) - (E_H - B_H)]$, where E_P is the value at the end of the peanut-supplemented diet period and B_P is the value just before the peanut-supplemented diet period. E_H is defined as the value at the end of the habitual diet period and B_H is defined as the value just before the habitual diet period.
‡*P* values are for one-sample Student's *t* test for changes during the habitual diet period.
§*P* values are for one-sample Student's *t* test for changes during the peanut-supplemented period.
‖*P* values are for one-sample Student's *t* test of the peanut effect.

calculated based on blood pressure, TC, LDL-C, HDL-C and TAG concentrations. In the present study, most of the variables used to calculate CHD did not change significantly. However, HDL-C increased significantly, and because HDL-C is a very strong inverse predictor of cardiovascular events[16], the estimated CHD risk was reduced.

When subjects began to supplement their usual diet with peanuts, their intake of MUFA, PUFA, fibre, plant protein, α-tocopherol, Cu, folate, Zn and Mg increased and the intake of animal protein decreased. These nutrient changes are similar to the dietary changes recommended to prevent CVD and other chronic diseases[17]. Despite daily peanut consumption (~ 77 g, or 1884 kJ of energy), no weight gain was observed in the study subjects. This may be due to several factors. During peanut consumption, subjects spontaneously commented on the high satiety value of the peanuts[18] and reported difficulties with consumption of habitual diet. In the present study, peanut displaced 12 % of the normal energy intake of the subjects, and these subjects inadvertently compensated by decreasing carbohydrate intake by 6 %. Another factor may be that faecal fat and energy loss is great with consumption of whole peanuts[19]. In the present study, nearly all of the subjects complained of increased frequency of daefecation and fatty stools. Finally, as shown in some studies, peanuts may enhance energy expenditure[20]. More studies are required to determine the long-term effects of peanut consumption on lipid profiles, metabolic changes and CHD risk in hypercholesterolaemic men. However, the data presented here suggest that it might be possible to reduce CHD risk factors using a single, simple intervention such as increasing peanut consumption in hypercholesterolaemic men. In summary, these results indicate that short-term peanut consumption in free-living hypercholesterolaemic men might favourably improve lipid profiles, AIP and CHD risk.

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